PALM INTRANET

Day : Wednesday
Date: 8/4/2004
Time: 12:07:20

Inventor Name Search

Enter the first few letters of the Inventor's Last Name.
Additionally, enter the first few letters of the Inventor's First name.

Last Name	First Name	
wolffe	alan	Search

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collingwood	trevor	Search

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 FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 19:40:06 ON 04 AUG 2004
L1          5190 S CHROMATIN (S) REMODEL?
L2       3956868 S PROTEIN
L3        354083 S FUSION OR CHIMER?
L4         93703 S L2(S) L3
L5           115 S L1 AND L4
L6            28 S L5 NOT PY>=2001
L7            14 DUP REM L6 (14 DUPLICATES REMOVED)
L8         28869 S METHYLASE OR DEMETHYLASE OR ACETYLASE OR DEACETYLASE
L9           306 S L8 (P) L4
L10          134 S L9 NOT PY>=2001
L11            0 S L10 AND L1
L12        15372 S "CHROMATIN STRUCTURE"
L13            4 S L10 AND L12
L14            4 DUP REM L13 (0 DUPLICATES REMOVED)
L15        62928 S "BINDING DOMAIN"
L16         5193 S L4 AND L15
L17           20 S L16 AND L1
L18           10 DUP REM L17 (10 DUPLICATES REMOVED)
L19            2 S L18 NOT PY>=2001
L20         5117 S L1 NOT "REGULATE TRANSCRIPTION"
L21          179 S L20 AND L15
L22           43 S L21 AND L12
L23           43 S L22 NOT PY>=20001
L24           18 S L22 NOT PY>=2001
L25           11 DUP REM L24 (7 DUPLICATES REMOVED)
L26            0 S WOLFFE/AU
L27            0 S WOLFFE/AU
L28           50 S SMITH/AU
L29            9 S WOLFE/AU
L30           53 S WOLFFE
L31            1 S L30 AND L1
L32            0 S WOLFFE/AU
L33            0 S "WOLFFE"/AU

=>
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L1             0 S "CHROMATIN REMODEL"
L2           226 S CHROMATIN (S) REMODEL
L3             0 S L2 (P) DNMT
L4             0 S L2 AND DNMT
L5          4670 S CHROMATIN (S) REMODEL?
L6             2 S L5 (P) DNMT
L7             2 DUP REM L6 (0 DUPLICATES REMOVED)
L8             7 S L5 AND DNMT
L9             4 DUP REM L8 (3 DUPLICATES REMOVED)
L10          572 S L5 AND (METHYLASE OR DEMETHYLASE OR ACETYLASE OR DEACETYLASE
L11           96 S L10 NOT PY>=2000
L12           45 DUP REM L11 (51 DUPLICATES REMOVED)
L13            2 S L12 AND METHYL
L14           21 S L2 AND METHYL?
L15            5 S L14 NOT PY>=2000
L16            2 DUP REM L15 (3 DUPLICATES REMOVED)

=> s chromatin (s) structure
L17       19475 CHROMATIN (S) STRUCTURE

=> s l17 (p) (DNMT or acetylase or methylase or deacetylase or demethylase or
methylation)
L18        1660 L17 (P) (DNMT OR ACETYLASE OR METHYLASE OR DEACETYLASE OR DEMETH
                YLASE OR METHYLATION)

=> s l18 not py>=2000
L19         762 L18 NOT PY>=2000

=> dup rem l19
PROCESSING COMPLETED FOR L19
L20         345 DUP REM L19 (417 DUPLICATES REMOVED)

=> s l20 and DNMT
L21           0 L20 AND DNMT
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L Number	Hits	Search Text	DB	Time stamp
1	1645	transcription WITH (modulator or modifier or alter or regulat)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:33
4	5116	chromatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:33
9	13715	nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
10	187	(nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
12	83	(((nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin) and "zinc finger") and "dna binding"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
16	0	(chromatin WITH remodel) SAME DNMT	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
2	44	(transcription WITH (modulator or modifier or alter or regulat)) SAME chromatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
3	5	6534261.pn. or 6607882.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
5	254	chromatin WITH remodeling	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
6	146	(chromatin WITH remodeling) and ("fusion protein" or "fusion construct")	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
7	137	((chromatin WITH remodeling) and ("fusion protein" or "fusion construct")) and (subunit or component or multi-protein or multiprotein)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
8	70	(((chromatin WITH remodeling) and ("fusion protein" or "fusion construct")) and (subunit or component or multi-protein or multiprotein)) and "zinc finger"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
11	93	((nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin) and "zinc finger"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
13	33	((((nurf or hoac or "swi/snf" or brm or brg or baf or chd2 or chd3 or chd4 or mot1 or rsc or HDAC or BAF or BRG1 or RSF) SAME chromatin) and "zinc finger") and "dna binding") and "chromatin structure"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
14	3	6607882.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
15	10	chromatin SAME DNMT	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34

17	29	chromatin WITH remodel	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
18	1	(chromatin WITH remodel) SAME (methylase or demethylase or acetylase or deacetylase or helicase)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:34
19	40880	"fusion protein" or "chimeric protein" or chimera or "fusion construct"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:35
20	1907	("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:35
21	758	(("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin) and "DNA binding domain"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:36
22	301	chromatin WITH remodel$	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:36
23	112	((("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin) and "DNA binding domain") and (chromatin WITH remodel$)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:36
26	991	"chromatin structure"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:37
27	61	((((("fusion protein" or "chimeric protein" or chimera or "fusion construct") and chromatin) and "DNA binding domain") and (chromatin WITH remodel$)) and "chromatin structure"	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/08/04 19:37

ANSWER 1 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN

ACCESSION NUMBER: 2000157824 EMBASE
TITLE: Lsh, an SNF2/helicase family member, is required for proliferation of mature T lymphocytes.
AUTHOR: Geiman T.M.; Muegge K.
CORPORATE SOURCE: K. Muegge, Lab. of Molecular Immunoregulation, Intramural Research Support Program, Frederick Can. Res. and Devt. Center, Frederick, MD 21702-1201, United States. muegge@mail.ncifcrf.gov
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (25 Apr 2000) 97/9 (4772-4777). Refs: 28
ISSN: 0027-8424 CODEN: PNASA6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Lsh (Hells) is closely related to SNF2/helicase family members that **remodel chromatin** and thus regulate gene transcription. In the adult mouse Lsh is expressed primarily in lymphoid tissue, showing the highest level in thymocytes. Lsh gene expression can be induced in thymic pro-T cells by pre-T cell receptor crosslinking and in mature T cells by T cell receptor crosslinking together with costimulation via CD28. The time course of Lsh gene and **protein** expression correlated closely with the onset of S phase of the cell cycle. To explore the function of Lsh during lymphoid development or activation, we deleted the Lsh gene by homologous recombination in ES cells. Fetal liver cells from Lsh-/- were used as a source of hematopoietic precursors to reconstitute lymphoid development in Rag2-/- mice. Lsh-/- (compared to Lsh+/+ or ±) **chimeras** showed a modest reduction in thymocyte numbers due to a partial arrest at the transition from the CD4-CD8- stage to the CD4+CD8+ stage of T cell development. Mature peripheral lymphocytes were reduced in number to .simeq.60% for T cells and 40% for B cells; however, V(D)J recombination of the immune receptor genes was normal. Although polyclonal activation of Lsh-/- T cells induced normal levels of cytokines, cell proliferation was severely suppressed and cells underwent apoptosis. Several genes involved in the regulation of apoptosis were expressed normally with the exception of Bcl-2 that was actually elevated. These findings demonstrate that Lsh is not obligatory for normal lymphoid development but is essential for normal proliferation of peripheral T lymphocytes.

L7 ANSWER 2 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:114294 BIOSIS
DOCUMENT NUMBER: PREV200100114294
TITLE: Dynamic binding of histone H1 to chromatin in living cells.
AUTHOR(S): Misteli, Tom [Reprint author]; Gunjan, Akash; Hock, Robert; Bustin, Michael; Brown, David T.
CORPORATE SOURCE: National Cancer Institute, NIH, Bethesda, MD, 20892, USA mistelit@mail.nih.gov
SOURCE: Nature (London), (14 December, 2000) Vol. 408, No. 6814, pp. 877-881. print.
CODEN: NATUAS. ISSN: 0028-0836.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Mar 2001
Last Updated on STN: 15 Feb 2002

AB The linker histone H1 is believed to be involved in chromatin organization by stabilizing higher-order chromatin structure. Histone H1 is generally viewed as a repressor of transcription as it prevents the access of

transcription factors and **chromatin remodelling** complexes to DNA. Determining the binding properties of histone H1 to chromatin in vivo is central to understanding how it exerts these functions. We have used photobleaching techniques to measure the dynamic binding of histone H1-GFP to unperturbed chromatin in living cells. Here we show that almost the entire population of H1-GFP is bound to chromatin at any one time; however, H1-GFP is exchanged continuously between chromatin regions. The residence time of H1-GFP on chromatin between exchange events is several minutes in both euchromatin and heterochromatin. In addition to the mobile fraction, we detected a kinetically distinct, less mobile fraction. After hyperacetylation of core histones, the residence time of H1-GFP is reduced, suggesting a higher rate of exchange upon **chromatin remodelling**. These results support a model in which linker histones bind dynamically to chromatin in a stop-and-go mode.

L7 ANSWER 3 OF 14 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001077962 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11129039
TITLE: The Arabidopsis PHD-finger protein SHL is required for proper development and fertility.
AUTHOR: Mussig C; Kauschmann A; Clouse S D; Altmann T
CORPORATE SOURCE: Max Planck Institut fur Molekulare Pflanzenphysiologie, Dept. Willmitzer, Potsdam, Germany.
SOURCE: Molecular & general genetics : MGG, (2000 Nov) 264 (4) 363-70.
Journal code: 0125036. ISSN: 0026-8925.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200101
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010111

AB The SHL gene from Arabidopsis thaliana encodes a small nuclear protein that contains a BAH domain and a PHD finger. Both domains are found in numerous (putative) transcriptional regulators and **chromatin-remodeling** factors. Different sets of transgenic lines were established to analyze the physiological relevance of SHL. SHL expression driven by the CaMV 35S promoter results in reduced growth, early flowering, early senescence, and impaired flower and seed formation. Antisense inhibition of SHL expression gives rise to dwarfism and delayed development. In-frame N-terminal **fusion** of the SHL **protein** to beta-glucuronidase (GUS) directs GUS to the nucleus of stably transformed Arabidopsis plants. Thus, SHL encodes a novel putative regulator of gene expression, which directly or indirectly influences a broad range of developmental processes.

L7 ANSWER 4 OF 14 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 1999421987 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10490642
TITLE: Leukemic HRX fusion proteins inhibit GADD34-induced apoptosis and associate with the GADD34 and hSNF5/INI1 proteins.
AUTHOR: Adler H T; Chinery R; Wu D Y; Kussick S J; Payne J M; Fornace A J Jr; Tkachuk D C
CORPORATE SOURCE: VA Puget Sound Health Care System, Seattle, Washington 98108, USA.
CONTRACT NUMBER: CA 68485 (NCI)
CA73969 (NCI)
SOURCE: Molecular and cellular biology, (1999 Oct) 19 (10) 7050-60.
Journal code: 8109087. ISSN: 0270-7306.

PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000209
Last Updated on STN: 20000209
Entered Medline: 20000203

AB One of the most common chromosomal abnormalities in acute leukemia is a reciprocal translocation involving the HRX gene (also called MLL, ALL-1, or HTRX) at chromosomal locus 11q23, resulting in the formation of HRX fusion proteins. Using the yeast two-hybrid system and human cell culture coimmunoprecipitation experiments, we show here that HRX proteins interact directly with the GADD34 protein. We have found that transfected cells overexpressing GADD34 display a significant increase in apoptosis after treatment with ionizing radiation, indicating that GADD34 expression not only correlates with apoptosis but also can enhance apoptosis. The amino-terminal third of the GADD34 protein was necessary for this observed increase in apoptosis. Furthermore, coexpression of three different HRX fusion proteins (HRX-ENL, HRX-AF9, and HRX-ELL) had an anti-apoptotic effect, abrogating GADD34-induced apoptosis. In contrast, expression of wild-type HRX gave rise to an increase in apoptosis. The difference observed here between wild-type HRX and the leukemic HRX fusion proteins suggests that inhibition of GADD34-mediated apoptosis may be important to leukemogenesis. We also show here that GADD34 binds the human SNF5/INI1 protein, a member of the SNF/SWI complex that can **remodel chromatin** and activate transcription. These studies demonstrate, for the first time, a gain of function for leukemic HRX **fusion** proteins compared to wild-type **protein**. We propose that the role of HRX fusion proteins as negative regulators of post-DNA-damage-induced apoptosis is important to leukemia progression.

L7 ANSWER 5 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1999396183 EMBASE
TITLE: Cloning and analysis of a Toxoplasma gondii histone acetyltransferase: A novel **chromatin remodelling** factor in Apicomplexan parasites.
AUTHOR: Hettmann C.; Soldati D.
CORPORATE SOURCE: D. Soldati, Zent. Molekulare Biologie Heidelberg, Im Neuenheimer Feld 282, 69120 Heidelberg, Germany. soldati@sun0.urz.uni-heidelberg.de
SOURCE: Nucleic Acids Research, (15 Nov 1999) 27/22 (4344-4352). Refs: 68
ISSN: 0305-1048 CODEN: NARHAD
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The yeast transcriptional adaptor GCN5 functions as a histone acetyltransferase, directly linking **chromatin** modification to transcriptional regulation. Homologues of yeast GCN5 have been found in Tetrahymena, Drosophila, Arabidopsis and human, suggesting that this pathway of **chromatin remodelling** is evolutionarily conserved. Consistent with this view, we have identified the Toxoplasma gondii homologue, referred to here as TgGCN5. The gene codes for a **protein** of 474 amino acids with an estimated molecular mass of 53 kDa. The **protein** reveals two regions of close similarity with the GCN5 family members, the HAT domain and the bromodomain. TgGCN5 occurs in a single copy in the T. gondii genome. The introduction of a second

copy of TgGCN5 in T. gondii tachyzoites is toxic unless the HAT activity is disrupted by a single point mutation. Full TgGCN5 does not complement the growth defect in a yeast gcn5- mutant strain, but a **chimera** comprising the T. gondii HAT domain fused to the remainder of yGCN5 does. These data show that T. gondii GNC5 is a histone acetyltransferase attesting to the significance of **chromatin remodelling** in gene regulation of Apicomplexa.

L7 ANSWER 6 OF 14 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1999403145 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10471746
TITLE: A conserved motif N-terminal to the DNA-binding domains of myogenic bHLH transcription factors mediates cooperative DNA binding with pbx-Meis1/Prep1.
AUTHOR: Knoepfler P S; Bergstrom D A; Uetsuki T; Dac-Korytko I; Sun Y H; Wright W E; Tapscott S J; Kamps M P
CORPORATE SOURCE: Department of Basic Science, Fred Hutchinson Cancer Research Center, Seattle, WA 98109, USA.
CONTRACT NUMBER: AG01228 (NIA)
AR45113 (NIAMS)
CA56876 (NCI)
SOURCE: Nucleic acids research, (1999 Sep 15) 27 (18) 3752-61. Journal code: 0411011. ISSN: 1362-4962.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199911
ENTRY DATE: Entered STN: 20000111
Last Updated on STN: 20010521
Entered Medline: 19991104
AB The t(1;19) chromosomal translocation of pediatric pre-B cell leukemia produces **chimeric** oncoprotein E2a-Pbx1, which contains the N-terminal transactivation domain of the basic helix-loop-helix (bHLH) transcription factor, E2a, joined to the majority of the homeodomain **protein**, Pbx1. There are three Pbx family members, which bind DNA as heterodimers with both broadly expressed Meis/Prep1 homeo-domain proteins and specifically expressed Hox homeodomain proteins. These Pbx heterodimers can augment the function of transcriptional activators bound to adjacent elements. In heterodimers, a conserved tryptophan motif in Hox proteins binds a pocket on the surface of the Pbx homeodomain, while Meis/Prep1 proteins bind an N-terminal Pbx domain, raising the possibility that the tryptophan-interaction pocket of the Pbx component of a Pbx-Meis/Prep1 complex is still available to bind trypto-phan motifs of other transcription factors bound to flanking elements. Here, we report that Pbx-Meis1/Prep1 binds DNA cooperatively with heterodimers of E2a and MyoD, myogenin, Mrf-4 or Myf-5. As with Hox proteins, a highly conserved tryptophan motif N-terminal to the DNA-binding domains of each myogenic bHLH family protein is required for cooperative DNA binding with Pbx-Meis1/Prep1. In vivo, MyoD requires this tryptophan motif to evoke **chromatin remodeling** in the Myogenin promoter and to activate Myogenin transcription. Pbx-Meis/Prep1 complexes, therefore, have the potential to cooperate with the myogenic bHLH proteins in regulating gene transcription.

L7 ANSWER 7 OF 14 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 1999315905 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10385516
TITLE: Large-scale **chromatin** unfolding and **remodeling** induced by VP16 acidic activation domain.
AUTHOR: Tumbar T; Sudlow G; Belmont A S
CORPORATE SOURCE: Program in Biophysics and Computational Biology, University

of Illinois, Urbana-Champaign, Urbana, Illinois 61801, USA.
CONTRACT NUMBER: R01-GM42516 (NIGMS)
SOURCE: Journal of cell biology, (1999 Jun 28) 145 (7) 1341-54.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990816
Last Updated on STN: 19990816
Entered Medline: 19990802

AB Analysis of the relationship between transcriptional activators and chromatin organization has focused largely on lower levels of chromatin structure. Here we describe striking **remodeling** of large-scale **chromatin** structure induced by a strong transcriptional activator. A VP16-lac repressor **fusion protein** targeted the VP16 acidic activation domain to chromosome regions containing lac operator repeats. Targeting was accompanied by increased transcription, localized histone hyperacetylation, and recruitment of at least three different histone acetyltransferases. Observed effects on large-scale **chromatin** structure included unfolding of a 90-Mbp heterochromatic chromosome arm into an extended 25-40-micrometers chromonema fiber, **remodeling** of this fiber into a novel subnuclear domain, and propagation of large-scale **chromatin** unfolding over hundreds of kilobase pairs. These changes in large-scale chromatin structure occurred even with inhibition of ongoing transcription by alpha-amanitin. Our results suggest a functional link between recruitment of the transcriptional machinery and changes in large-scale chromatin structure. Based on the observed long-range propagation of changes in large-scale chromatin structure, we suggest a possible rationale for the observed clustering of housekeeping genes within Mbp-sized chromosome bands.

L7 ANSWER 8 OF 14 MEDLINE on STN
ACCESSION NUMBER: 1999326293 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10397708
TITLE: **Chromatin remodeling** and leukemia: new therapeutic paradigms.
AUTHOR: Redner R L; Wang J; Liu J M
CORPORATE SOURCE: Division of Hematology/Oncology, Department of Medicine, University of Pittsburgh/Medical Center, Pittsburgh, PA, USA.. redner+@pitt.edu
CONTRACT NUMBER: CA67346 (NCI)
SOURCE: Blood, (1999 Jul 15) 94 (2) 417-28. Ref: 149
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990816
Last Updated on STN: 19990816
Entered Medline: 19990805

L7 ANSWER 9 OF 14 EMBASE
on STN
ACCESSION NUMBER: 1998320304 EMBASE
TITLE: Subunits of the yeast SWI/SNF complex are members of the actin-related protein (ARP) family.
AUTHOR: Peterson C.L.; Zhao Y.; Chait B.T.
CORPORATE SOURCE: C.L. Peterson, UMMC, Biotech 2, 373 Plantation St.,

Worcester, MA 01605, United States.
craig.peterson@ummed.edu
SOURCE: Journal of Biological Chemistry, (11 Sep 1998) 273/37 (23641-23644).
Refs: 35
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The yeast SWI/SNF **chromatin remodeling** complex is comprised of 11 tightly associated polypeptides (SWI1, SWI2, SWI3, SNF5, SNF6, SNF11, SWP82, SWP73, SWP59, SWP61, and SWP29). We have used matrix-assisted laser desorption ionization time-of-flight mass spectrometry to identify the genes that encode the SWP59 and SWP61 subunits. Surprisingly, we find that SWP59 and SWP61 are encoded by the ARP9 and ARP7 genes, respectively, which encode members of the actin-related **protein** (ARP) family. Sequence analyses have shown that ARP9 and ARP7 are 24-26% identical (48-51% similar) to yeast actin and that they are likely to maintain the overall actin fold. Deletion of either the ARP9 or ARP7 gene causes typical swi/snf phenotypes, including growth defects on media containing galactose, glycerol, or sucrose as sole carbon sources. ARP9 and ARP7 are also required for expression of an HO-lacZ **fusion** gene and for full transcriptional enhancement by the GAL4 activator. The identification of two ARP family members as crucial subunits of the SWI/SNF complex suggests that the complex may contain a total of three different ATPase subunits; furthermore, the similarity of ARP7 and ARP9 to the HSP and HSC family of ATPases suggests the possibility that **chromatin remodeling** by SWI/SNF may involve chaperone-like activities.

L7 ANSWER 10 OF 14 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 1998187598 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9528749
TITLE: SWI-SNF complex participation in transcriptional activation at a step subsequent to activator binding.
AUTHOR: Ryan M P; Jones R; Morse R H
CORPORATE SOURCE: Molecular Genetics Program, Wadsworth Center, New York State Department of Health, and State University of New York School of Public Health, Albany 12201-2002, USA.
CONTRACT NUMBER: GM51993 (NIGMS)
SOURCE: Molecular and cellular biology, (1998 Apr) 18 (4) 1774-82.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980529
Last Updated on STN: 19980529
Entered Medline: 19980521
AB The SWI-SNF complex in yeast and related complexes in higher eukaryotes have been implicated in assisting gene activation by overcoming the repressive effects of chromatin. We show that the ability of the transcriptional activator GAL4 to bind to a site in a positioned nucleosome is not appreciably impaired in swi mutant yeast cells. However, **chromatin remodeling** that depends on a transcriptional activation domain shows a considerable, although not complete, SWI-SNF dependence, suggesting that the SWI-SNF complex exerts its major effect at a step subsequent to activator binding. We tested this idea further by comparing the SWI-SNF dependence of a reporter gene based on the GAL10 promoter, which has an accessible upstream activating

sequence and a nucleosomal TATA element, with that of a CYC1-lacZ reporter, which has a relatively accessible TATA element. We found that the GAL10-based reporter gene showed a much stronger SWI-SNF dependence than did the CYC1-lacZ reporter with several different activators. Remarkably, transcription of the GAL10-based reporter by a GAL4-GAL11 **fusion protein** showed a nearly complete requirement for the SWI-SNF complex, strongly suggesting that SWI-SNF is needed to allow access of TFIID or the RNA polymerase II holoenzyme. Taken together, our results demonstrate that **chromatin remodeling** in vivo can occur by both SWI-SNF-dependent and -independent avenues and suggest that the SWI-SNF complex exerts its major effect in transcriptional activation at a step subsequent to transcriptional activator-promoter recognition.

L7 ANSWER 11 OF 14 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 1999110097 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9894806
TITLE: Recruitment of the RNA polymerase II holoenzyme and its implications in gene regulation.
AUTHOR: Barberis A; Gaudreau L
CORPORATE SOURCE: Institute of Molecular Biology, University of Zurich, Switzerland.
SOURCE: Biological chemistry, (1998 Dec) 379 (12) 1397-405. Ref: 82
Journal code: 9700112. ISSN: 1431-6730.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990326
Last Updated on STN: 19990326
Entered Medline: 19990317

AB In yeast cells, interaction between a DNA-bound protein and a single component of the RNA polymerase II (poIII) holoenzyme is sufficient to recruit the latter to a promoter and thereby activate gene transcription. Here we review results which have suggested such a simple mechanism for how genes can be turned on. The series of experiments which eventually led to this model was originally instigated by studying gene expression in a yeast strain which carries a point mutation in Gal11, a component of the poIII holoenzyme. In cells containing this mutant protein termed Gal11P, a derivative of the transcriptional activator Gal4 devoid of any classical activating region is turned into a strong activator. This activating function acquired by an otherwise silent DNA-binding protein is solely due to a novel and fortuitous interaction between Gal11P and a fragment of the Gal4 dimerization region generated by the P mutation. The simplest explanation for these results is that tethering Gal11 to DNA recruits the poIII holoenzyme and, consequently, activates gene transcription. Transcription factors that are believed not to be integral part of the poIII holoenzyme but are nevertheless required for this instance of gene activation, e.g. the TATA-binding TFIID complex, may bind DNA cooperatively with the holoenzyme when recruited to a promoter, thus forming a complete poIII preinitiation complex. One prediction of this model is that recruitment of the entire poIII transcription complex and consequent gene activation can be achieved by tethering different components to DNA. Indeed, **fusion** of a DNA-binding domain to a variety of poIII holoenzyme components and TFIID subunits leads to activation of genes bearing the recognition site for the DNA-binding **protein**. These results imply that accessory factors, which are required to remove or modify nucleosomes do not need to be directly contacted by activators, but can rather be engaged in the activation

process when the poIII complex is recruited to DNA. In fact, recruitment of the poIII holoenzyme suffices to remodel nucleosomes at the PHO5 promoter and presumably at many other promoters. Other events in the process of gene expression following recruitment of the transcription complex, e.g. initiation, promoter clearance, elongation and termination, could unravel as a consequence of the recruitment step and the formation of an active preinitiation complex on DNA. This view does not exclude the possibility that classical activators also act directly on **chromatin remodeling** and post-recruitment steps to regulate gene expression.

L7 ANSWER 12 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1999396668 EMBASE
TITLE: Cytoplasmic control of nuclear assembly.
AUTHOR: Collas P.
CORPORATE SOURCE: P. Collas, University of Oslo, Institute of Medical Biochemistry, PO Box 1112 Blindern, N-0317 Oslo, Norway. philippe.collas@basalmed.uio.no
SOURCE: Reproduction, Fertility and Development, (1998) 10/7-8 (581-592).
Refs: 61
ISSN: 1031-3613 CODEN: RFDEEH
COUNTRY: Australia
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 021 Developmental Biology and Teratology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The reconstitution of a replication-competent, transcriptionally active nucleus following mitosis, fertilization or nuclear transplantation involves a stepwise series of reactions, most (if not all) of which are controlled by the cytoplasmic environment. This review discusses the nature of cytoplasmic contributions to the development of the male pronucleus at fertilization, and the effect of altering the cytoplasmic environment on nuclear assembly. The system used to investigate these regulations consists of permeabilized sea urchin sperm nuclei incubated under controlled conditions in a cell-free extract of fertilized sea urchin eggs. (1) In egg cytoplasmic extract, male pronuclear formation is initiated by the disassembly of the sperm nuclear lamina as a result of lamin phosphorylation by a cytosolic **protein** kinase C. (2) Sperm histones are phosphorylated by an as yet unidentified soluble kinase. (3) The conical sperm nucleus decondenses into a spherical pronucleus in an ATP-and cytosolic pH-dependent manner. (4) **Chromatin** decondensation is associated with the replacement of sperm histones by maternal histones. (5) Nuclear membranes form by ATP-dependent binding of vesicles to **chromatin** and GTP-dependent **fusion** of these vesicles to one another. (6) Three cytoplasmic vesicle populations with distinct biochemical, **chromatin**-binding and **fusion** properties are required for nuclear envelope assembly. (7) Targeting of the bulk of nuclear membrane vesicles to **chromatin** is mediated by an integral membrane **protein** similar to human lamin B receptor. (8) The last step of male pronuclear formation, nuclear swelling, is promoted by the assembly of nuclear pores, nuclear import of soluble lamins and growth of the nuclear membranes. (9) Once inside the nucleus, lamin B associates with lamin B receptors, presumably to tether the inner nuclear membrane with the lamina. Overall, these processes are similar to those characterizing nuclear reconstitution after mitosis in somatic cells or nuclear **remodeling** following transplantation into oocytes or eggs. The influence of the egg cytoplasmic environment on some aspects of nuclear **remodeling** after nuclear transplantation is also discussed.

L7 ANSWER 13 OF 14 MEDLINE on STN DUPLICATE 7

ACCESSION NUMBER: 1998259288 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9597000
TITLE: Cellular localization and expression of template-activating factor I in different cell types.
AUTHOR: Nagata K; Saito S; Okuwaki M; Kawase H; Furuya A; Kusano A; Hanai N; Okuda A; Kikuchi A
CORPORATE SOURCE: Department of Biomolecular Engineering, Faculty of Bioscience and Biotechnology, Tokyo Institute of Technology, Yokohama, Japan.
SOURCE: Experimental cell research, (1998 May 1) 240 (2) 274-81. Journal code: 0373226. ISSN: 0014-4827.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980618
Last Updated on STN: 19980618
Entered Medline: 19980608

AB Template-activating factors I (TAF-I) alpha and beta have been identified as **chromatin remodeling** factors from human HeLa cells. TAF-I beta corresponds to the **protein** encoded by the set gene, which was found in an acute undifferentiated leukemia as a **fusion** version with the can gene via chromosomal translocation. To determine the localization of TAF-I, we raised both polyclonal and monoclonal antibodies against TAF-I. The proteins that react to the antibodies are present not only in human cells but also in mouse, frog, insect, and yeast cells. The mouse TAF-I homologue is ubiquitous in a variety of tissue cells, including liver, kidney, spleen, lung, heart, and brain. It is of interest that the amounts of TAF-I alpha and beta vary among hemopoietic cells and some specific cell types do not contain TAF-I alpha. The level of the TAF-I proteins does not change significantly during the cell cycle progression in either HeLa cells synchronized with an excess concentration of thymidine or NIH 3T3 cells released from the serum-depleted state. TAF-I is predominantly located in nuclei, while TAF-I that is devoid of its acidic region, the region which is essential for the TAF-I activity, shows both nuclear and cytoplasmic localization. The localization of TAF-I in conjunction with the regulation of its activity is discussed.

L7 ANSWER 14 OF 14 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 97093330 EMBASE
DOCUMENT NUMBER: 1997093330
TITLE: Interaction of a Swi3 homolog with Sth1 provides evidence for a Swi/Snf- related complex with an essential function in Saccharomyces cerevisiae.
AUTHOR: Treich I.; Carlson M.
CORPORATE SOURCE: M. Carlson, HSC922, 701 W. 168th St., New York, NY 10032, United States
SOURCE: Molecular and Cellular Biology, (1997) 17/4 (1768-1775). Refs: 61
ISSN: 0270-7306 CODEN: MCEBD4
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The Saccharomyces cerevisiae Swi/Snf complex has a role in **remodeling chromatin** structure to facilitate transcriptional activation. The complex has 11 components, including Swi1/Adr6, Swi2/Snf2, Swi3, Snf5, Snf6, Snf11, Swp73/Snf12, and Tfg3. Mammalian homologs of these proteins have been shown to form multiple Swi/Snf-related complexes. Here we characterize an S. cerevisiae Swi3

homolog (Swh3) and present evidence that it associates in a complex with a Snf2 homolog, Sth1. We identified Swh3 as a **protein** that interacts with the N terminus of Snf2 in the two-hybrid system. Swh3 and Swi3 are functionally distinct, and overexpression of one does not compensate for loss of the other. Swh3 is essential for viability and does not activate transcription of reporters. The Snf2 sequence that interacts with Swh3 was mapped to a region conserved in Sth1. We show that Swh3 and Sth1 **fusion** proteins interact in the two-hybrid system and coimmunoprecipitate from yeast cell extracts. We also map interactions between Swh3 and Sth1 and examine the role of a leucine zipper motif in self-association of Swh3. These findings, together with previous analysis of Sth1, indicate that Swh3 and Sth1 are associated in a complex that is functionally distinct from the Swi/Snf complex and essential for viability.